SARCOIDOSIS VASCULITIS AND DIFFUSE LUNG DISEASES 2025; 42 (2): 15884 DOI: 10.36141/svdld.v42i2.15884

Hypersensitivity pneumonitis in Türkiye: An underrecognized pulmonary disorder

Oguz Uzun[1], Busra Adıguzel Gundogdu[1], Yurdanur Sullu[2], Kader Topçu Erturk[1], Sümeyye Kement[1], Fatma Selen Ala Cıtlak[1]

[1]Ondokuz Mayis University, Faculty of Medicine, Department of Pulmonary Medicine, Samsun, Turkey; [2]Ondokuz Mayis University, Faculty of Medicine, Department of Pathology, Samsun, Turkey

Abstract. *Introduction:* The aims of this study are to investigate hypersensitivity pneumonitis (HP) related publications from Türkiye, to discuss features of HP concerning our case series, and to evaluate our patients in terms of diagnostic confidence according to existing guidelines. *Methods:* We conducted a comprehensive review of the literature to analyse cumulated data about HP publications originating from Türkiye. Additionally, we evaluated HP cases diagnosed and followed in last 5 year at our hospital. *Results:* A total of 34 HP-related publications originated from Türkiye. Thirteen articles reported pediatric cases, while 20 articles included adult patients. Bird fancier's disease was the most common etiology of HP. A total of 138 patients were included in this study. Of these, 107 (77.5%) were female, and 31 (22.5%) were male. According to the American Thoracic Society (ATS) 2020 guidelines, 45 patients (33%) had a definite diagnosis, whereas 94 patients (68%) were diagnosed as definite cases according to the American College of Chest Physician's (ACCP) 2021 guidelines. Eighty-five patients (62%) had the non-fibrotic form, while 53 patients (38%) had the fibrotic form. The most frequent etiology was barn located beneath houses in rural areas (58 patients, 42%). *Conclusion:* This study highlights that HP, although underrepresented in Turkish medical literature, is more prevalent than previously recognized. Barns located beneath houses in rural areas and moldy environments in urban settings are the two main etiologies of HP in our region. The high prevalence of chronic cases in our patient group highlights the difficulty of diagnosing HP at early stages. We propose refining HP classification into "chronic inflammatory" and "chronic fibrotic" subtypes and advocate for a pragmatic diagnostic approach that avoids unnecessary invasive procedures in patients with identifiable antigens and characteristic HRCT findings.

Key words: hypersensitivity pneumonitis, high-resolution computed tomography, mosaic attenuation, farmer's lung, indoor air pollution

Background

Hypersensitivity pneumonitis (HP) is an inflammatory interstitial lung disease caused immune reactions to various antigen. It frequently goes unrecognized and is often misdiagnosed as respiratory infections in its acute and subacute forms or as idiopathic pulmonary fibrosis (IPF) in its chronic form. The prevalence of HP varies depending on factors such as diagnostic criteria, environmental and genetic influences, host susceptibility, and geographic conditions (1-5). It is estimated to be 0.3 and 0.9 per 100,000 individuals, even higher values have been reported for bird breeder's disease (6). In a multicenter study conducted in Türkiye, HP accounted for 4% of all interstitial lung disease (ILD) cases (3). However, more recent studies and registries suggest increasing prevalence rates, with reports as high as 47% (7-9).

Received: 29 March 2024
Accepted: 2 January 2025
Correspondence: Dr. Oğuz Uzun
Ondokuz Mayıs Üniversitesi, Tıp Fak., Göğüs Hst. AD 555139 Samsun/Türkiye
E-mail: oguzuzun67@yahoo.com.tr
Orcid ID: 0000-0002-0475-4198

The diagnosis of HP remains challenging due to overlapping clinical, radiological, and pathological findings with other ILDs, particularly IPF (10). Despite its significance, HP remains underrepresented in academic literature in Türkiye. The objectives of this study are to evaluate HP-related publications from Türkiye, analyze clinical and diagnostic characteristics of HP cases in our institution, and compare diagnostic confidence using the American Thoracic Society (ATS) 2020 and American College of Chest Physician's (ACCP) 2021 guidelines (4,5).

Materials and methods

Review of Türkiye-Originated HP literature

We conducted a comprehensive search of PubMed, Google, and Turkish Medline using keywords "hypersensitivity pneumonitis," "extrinsic allergic alveolitis," and their Turkish equivalents ("hipersensitivite pnömonisi" and "ekstrensek alerjik alveolit"). Articles in English, Turkish, and German were included. Relevant references cited in these articles were also reviewed.

HP cases from our institution

We analyzed HP cases diagnosed and followed over the past five years at our tertiary referral center, which has expertise in interstitial lung diseases, radiology, and pathology.

Data collection included:

1. Demographic and clinical characteristics of the HP cases
2. Underlying etiologies
3. Radiological and pathological findings
4. Clinic presentation and classification based on consensus guideline
5. Diagnostic confidence according to ATS and ACCP guidelines

Ethical approval was obtained from the Institutional Ethics Committee (Ondokuz Mayıs University, 2022/584), and all participants provided informed consent. Statistical analyses were conducted using appropriate methods, with data presented as means with standard deviations or frequencies as percentage.

Results

Characteristics of our HP cases

We included 138 HP patients, of whom 107 (77.5%) were female, and 31 (22.5%) were male. The mean age was 64 years old (65 for females and 61 for males). Non-fibrotic cases had a mean age of 60 years, while fibrotic cases had a mean age of 70 years. Chronic patients were significantly older than acute and subacute cases. At the time of diagnosis, 116 (84%) of the patients were non-smokers, 17 (12%) of the patients were ex-smokers and 5 (4%) were active smokers. A definite diagnosis has been made to 45 (33%) patients according to the ATS guideline and to 94 (68%) patients according to the ACCP guideline. The diagnostic classification and confidence according to ATS and ACCP guidelines are shown on Table 1.

The clinical presentations of the patients were acute in 20, subacute in 58, and chronic in 110 patients according to the original classification of HP. The presentation of the patients according to classic and new ATS and ACCP guidelines is shown in Table 2.

Table 1. Diagnostic confidence according to ATS 2020 and ACCP 2021 Guidelines

ATS 2020			**ACCP 2021**		
	Frequency	**Percent**		**Frequency**	**Percent**
Definite (> 90%)	45	33	Definite HP	94	68
High confidence (80-89%)	6	4	Provisional high confidence	7	5
Moderate confidence (70-79%)	49	35.5			
Low confidence (51-69%)	23	17	Provisional low confidence	34	27
Not excluded	15	11	HP unlikely	3	2
Total	138	100.0	Total	138	100

Table 2. Comparison of Hypersensitivity Pneumonitis (HP) Classifications in Published Cohorts

Classification System	Category	Number of Patients (%)
Original Classification	Acute	20 (14.5%)
	Subacute	58 (42%)
	Chronic	60 (43.5%)
Vasakova et al., 2017 (43)	Acute	20 (14%)
	Chronic	118 (85.5%)
	—Inflammatory	65 (47%)
	— Fibrotic	53 (38%)
ATS 2020 / ACCP 2021 (4–5)	Non-fibrotic	85 (62%)
	Fibrotic	53 (38%)

Table 3. HRCT findings of the patients

Radiological Classification	Number (percent)
Typical HP	96 (70)
Compatible with HP	33 (24)
Indeterminate for HP	8 (6)
Total	138 (100)

All patients underwent HRCT. Typical HP findings were observed in 70% of patients, while 24% were classified as compatible with HP and 6% as indeterminate (Table 3).

Bronchoscopy was performed on 85 patients, and surgical biopsies on six patients. Since cryobiopsy is not available in our institution, we have not used this method on any patient. No severe complications like severe haemorrhage or death occurred due to these procedures. Pneumothorax has occurred after the transbronchial biopsy in two patients who were successfully treated with closed tube thoracostomy underwater drainage. Bronchoalveolar lavage (BAL) was performed on 89 patients. Lymphocytic BAL (>30%) was observed in 43% of the cases. The results of lung biopsies according to the ATS 2020 guideline are shown in Table 4. The results of the BAL and/or biopsy procedure did not alter the diagnoses of HP in any of the patients. However, they did modify the diagnostic probabilities as follows: in 14 patients, the classification changed from "definite HP" to "high confidence HP"; in 5 patients, from "high confidence HP" to "moderate confidence HP"; in 3 patients, from "definite HP" to "moderate confidence HP"; in 1 patient, from "moderate" to "low confidence HP"; in 1 patient from "moderate" to "not-excluded HP"; and in 1 patient, from "high" to "low confidence HP".

Table 4. Pathological Results of 85* HP Patients according to ATS 2020 Guideline

Pathologic Result of Biopsy	Number (percent)
Typical HP	49 (58)
Probable HP	3 (3.5)
Indeterminate for HP	3 (3.5)
Inadequate material	30 (35)
Total	85 (100)

*Both bronchoscopic transbronchial and surgical biopsy was performed in 5 patients.

Table 5. Identified Exposure Types of Our HP Patients

Type of exposure	Number (percent)
Barn	58 (42)
Moldy house	31 (22.5)
Birds Canary Pigeon Parrot Partridge	26 (19) 11 9 4 2
Poeltry	5 (4)
Naphthalene	1 (0.7)
Fertilizer	1 (0.7)
Undefined	16 (12)
Total	138 (100)

The most frequent etiology of our patient group was barn ("farmer's lung") in 58 (42%) patients. Other frequent underlying etiologies were moldy living environment ("indoor-air alveolitis") in 31 (22.5%) and birds in 26 (19%) patients (Table 5). The duration of exposure was more than 3 years in 116 (84%) patients.

Discussion

A total of 33 publications [28 case reports (11-38), five original articles (2, 39-41), and one review (42)] on HP from Türkiye were identified. Among these, 13 studies focused on pediatric cases, while 19 involved adult patients. Bird exposure (e.g., pigeon breeder's disease) was the leading etiology in these studies (Table 6).

The low number of HP-related publications in Türkiye (to our knowledge only 52 cases have been

Table 6. The properties of Türkiye-originated HP-related publications*

	Pediatric	Adult
Number of Türkiye- based Studies	9	20
Number of Original Articles	1	2
Review		1
Total Number of Cases Reported	34	18
Female/Male	16/18	8/9
Mean Age (F/M)	10 (11/8)	48,5 (49/36)
Acute/subacute/chronic	4/23/7	2/7/6**
Etiology of HP		
Pigeon	25	5
Budgerigar		2
Bird	1	2
Archive Dust		2
Straw mould		1
Hazelnut Farmer		1
Broom grass		1
Hard cash		1
Mushroom		1
Total	26	16

*Our institution does not see pediatric interstitial or HP cases. **3 patients are unknown.

reported, Table 1) highlights a lack of awareness and academic interest among physicians regarding this condition. Given Türkiye's humid climate and the high prevalence of agricultural and livestock activities, we anticipated a higher HP prevalence in the country. The limited awareness among physicians may be attributed to HP's rarity and its underrepresentation in medical education and residency training. Furthermore, diagnosing HP is inherently challenging due to its nonspecific clinical, radiological, and pathological features, which may lead to misdiagnosis, overlooked cases, or delayed diagnoses after irreversible changes have occurred (43). This is supported by the fact that 53 (38%) of our patients presented in the chronic fibrotic phase. However, multiple factors, including exposure-related variables (e.g., concentration, duration, frequency, particle size, and solubility), environmental conditions, and genetic predisposition, can influence HP's clinical course (4, 44-47). While HP is generally considered an adult disease, the notable number of pediatric cases in this study (Table 1) is intriguing. This raises the question of whether HP is genuinely more prevalent in pediatric populations or whether the finding reflects the presence of dedicated pediatric specialists. To clarify this, further registry studies focusing on interstitial lung diseases (ILD) and HP in pediatric populations are needed. It is important to note that pediatric HP cases have historically been considered exceedingly rare. HP develops in susceptible individuals following repeated exposure to various antigens. Hundreds of different sources of antigens have been reported (45). Numerous antigenic sources have been identified, varying across geographic regions, climates, occupational settings, and local habits. For example, in a Portuguese cohort (48), birds were the most frequent antigen source (76.6%), whereas household mold (48.8%) predominated in a South Korean cohort (49), and air coolers and conditioners (69.5%) were most common in an Indian cohort (9). In our cohort, contaminated barns (42%) and household mold (22.5%) emerged as the leading causes of HP. This finding reflects the housing conditions in rural areas, where barns are often located on the ground floors of homes, resulting in close human-livestock proximity. In urban settings, water leaks, aging infrastructure, and poor construction quality contribute to mold growth, causing what is often referred to as "indoor air alveolitis." These results emphasize the importance of public health education and preventive measures, as HP etiologies such as mold exposure and barn contamination are largely preventable. Our study, comprising 138 HP cases from a single center, underscores the significance of HP among ILDs in Türkiye. Although HP accounted for only 82 cases (4%) of the total 2245 patients reported in the 2014 ILD registry study (3), our findings suggest increased awareness and recognition of HP in recent years. However, diagnosing HP remains a major clinical challenge, as it often mimics other ILDs. HP should be considered in the differential diagnosis of most interstitial lung diseases. Nevertheless, validated diagnostic criteria are lacking, and diagnosis typically relies on a combination of antigen exposure history and compatible clinical, radiological, and pathological findings. The identification of the causative antigen remains a critical diagnostic step but is often difficult for clinicians. Consequently, HP is frequently misdiagnosed or overlooked, particularly in acute and subacute cases that mimic infections, or in chronic fibrotic cases mistaken for idiopathic

pulmonary fibrosis (IPF) or nonspecific interstitial pneumonia (NSIP). Historically, HP has been categorized into acute, subacute, and chronic phases. However, recent guidelines by ATS (4) and ACCP (5), as well as Vasakova et al. (43), propose a revised classification into "acute/inflammatory HP" and "chronic/fibrotic HP." In our study, 118 patients were classified as chronic HP based on symptom duration exceeding six months (Table 3). Of these, 53 (45%) were categorized as "chronic fibrotic" HP based on clinical, radiological, and pathological findings, while 65 (55%) exhibited "inflammatory features". Therefore, we suggest further subdividing chronic cases into "chronic inflammatory" and "chronic fibrotic" subtypes to better reflect disease heterogeneity (Table 3). The determinants of progression to chronic fibrotic disease remain unclear, though continuous low-level antigen exposure combined with genetic susceptibility is thought to play a key role. This hypothesis is supported by the significant proportion of fibrotic cases in our cohort, where patients experienced persistent, low-level exposure to mold or barn-related antigens. Further, as chronic fibrotic disease does not always follow episodes of disease, a time-based classification of HP may be misleading and does not capture the heterogeneity of disease. Since HP does not have a single definitive diagnostic method, ATS and ACCP have proposed an algorithm with diagnostic probabilities in their recently published guidelines (4-5). The ATS and ACCP guidelines propose diagnostic algorithms based on clinical, radiological, and pathological features to determine HP likelihood. We evaluated our patients' diagnostic probability according to these new guidelines (Table 2). In our cohort, significant discrepancies were observed between the two algorithms. For example, 94 patients (68%) had a definite HP diagnosis per ATS criteria, compared to only 45 patients (33%) per ACCP criteria. This difference likely stems from ATS guidelines requiring bronchoalveolar lavage (BAL) lymphocytosis and/or histopathologic confirmation, whereas ACCP guidelines allow a definite diagnosis based solely on antigen exposure and characteristic HRCT findings. Given that the results of the BAL and/or biopsy procedures did not modify the diagnoses of HP in any of the patient groups, we propose that bronchoscopy may be unnecessary in patients with identified inciting antigens and typical HRCT findings. Additionally, the limitations associated with invasive procedures, such as BAL and transbronchial biopsy—including patient reluctance, resource availability, and potential procedural complications—should also be acknowledged. In our cohort, adequate biopsy specimens were not obtained in 30 (35%) of the 85 patients who underwent bronchoscopy (Table 5). This aligns with recent IPF guidelines (50), which discourage surgical lung biopsy in patients with typical clinical and HRCT features compared to IPF guidelines 20 years ago (51). A similar approach may be appropriate for HP to minimize unnecessary procedures. Many invasive procedures can be avoided with this algorithm, such as 96 (70%) for our patient group. Additionaly, it has also been shown that HRCT can discriminate HP from other ILDs with a diagnostic accuracy of 85% (52). It should also be noted that HP has been widely recognized with the use of HRCT criteria alone, to the point that some countries, such as India, now recognize this as one of the most common causes of ILDs (50).

Although our study encompasses a considerable number of cases of this rare disease, it has several limitations. The study was conducted at a single center with a retrospective design, and no standardized diagnostic algorithm was utilized for patient diagnosis. In conclusion, this study highlights that HP, although underrepresented in Turkish medical literature, is more prevalent than previously recognized. Contaminated barns in rural areas and mold exposure in urban settings are the primary etiological factors. The predominance of chronic forms in our cohort underscores the need for early diagnosis and intervention. Enhanced education of both physicians and the public is critical to prevent diagnostic delays and improve outcomes. Finally, we propose refining HP classification into "chronic inflammatory" and "chronic fibrotic" subtypes and advocate for a pragmatic diagnostic approach that avoids unnecessary invasive procedures in patients with identifiable antigens and characteristic HRCT findings.

Authors' Contribution: O.U. and B.A.G. contributed to the study conception and design; material preparation, data collection, and analysis were performed by all authors; the first draft was written by O.U.; all authors commented on previous versions, read, and approved the final manuscript.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article

References

1. Fernández Pérez ER, Kong AM, Raimundo K, et al. Epidemiology of hypersensitivity pneumonitis among an insured population in the United States: a claims-based cohort analysis. Ann Am Thorac Soc. 2018;15(4):460–9. doi: 10.1513/annalsats.201704-288oc
2. Uzun O, Yilmaz K, Sunter AT, et al. The prevalence of "pigeon breeder's disease" in a Turkish city and review of the literature. Lung. 2011;189(3):243–50. doi: 10.1007/s00408-011-9292-5
3. Musellim B, Okumus G, Uzaslan E, et al. Epidemiology and distribution of interstitial lung diseases in Turkey. Clin Respir J. 2014;8(1):55–62. doi: 10.1111/crj.12035
4. Raghu G, Remy-Jardin M, Ryerson CJ, et al. Diagnosis of hypersensitivity pneumonitis in adults: an official ATS/JRS/ALAT clinical practice guideline. Am J Respir Crit Care Med. 2020;202(3): e36–e69. doi: 10.1164/rccm.202005-2032st
5. Fernández Pérez ER, Travis WD, Lynch DA, et al. Diagnosis and evaluation of hypersensitivity pneumonitis: CHEST guideline and expert panel report. Chest. 2021;160(2):e97–e156. doi: 10.1016/j.chest.2021.03.066
6. Morell F, Villar A, Ojanguren I, et al. Hypersensitivity pneumonitis and (idiopathic) pulmonary fibrosis due to feather duvets and pillows. Arch Bronconeumol. 2021;57(2):87–93. doi: 10.1016/j.arbres.2019.12.003
7. Morell F, Roger A, Reyes L, et al. Bird fancier's lung: a series of 86 patients. Medicine (Baltimore). 2008;87(2):110–30. doi: 10.1097/md.0b013e31816c3d3e
8. Karakatsani A, Papakosta D, Rapti A, et al. Epidemiology of interstitial lung diseases in Greece. Respir Med. 2009;103(8):1122–9. doi: 10.1016/j.rmed.2009.02.008
9. Singh S, Collins BF, Sharma BB, et al. Interstitial lung disease (ILD) in India: results of a prospective registry. Am J Respir Crit Care Med. 2017;195(6):801–13. doi: 10.1164/rccm.201607-1484oc
10. Morell F, Villar A, Montero MA, et al. Chronic hypersensitivity pneumonitis in patients diagnosed with idiopathic pulmonary fibrosis: a prospective case-cohort study. Lancet Respir Med. 2013;1(9):685–94. doi: 10.1016/s2213-2600(13)70164-4
11. Erkan F, Baur X, Kılıçarslan Z, et al. Exogene allergische alveolitis durch schimmelige Haselnusshüllblätter. Pneumologie. 1992;46: 32–5. PMID: 37878257
12. Göçmen A, Kiper N, Misirligil Z, et al. Hypersensitivity pneumonitis: a case with pigeon breeder's disease. J Pediatr Dis. 1992;35:29–35. PMID: 9433157
13. Erkan F, Ozyaral O, Baur X, et al. Extrinsic allergic alveolitis of housewives induced by indoor moulds. Eur Respir J. 1994; 7(Suppl 18):218s
14. Görgüner M, Mirici A, Ciftcioglu A, et al. Bird breeder's lung. Respiration. 1995;62:799–804
15. Dilber E, Ozcelik U, Gocmen A, et al. Recurrent pneumonitis in a 10-year-old girl with pigeon breeder's disease. Turk J Pediatr. 1997;39:541–5. PMID: 9433157
16. Karnak D, Kayacan O, Beder S, et al. Hypersensitivity pneumonitis and inhaled steroid. Tuberk Toraks. 2001;49(1):142–6
17. Yilmaz S, Cimrin A, Itil O, et al. Bird fancier's lung: clinical and pathological view. Turk Respir J. 2002;3:102–5
18. Yalcin E, Kiper N, Gocmen A, et al. Pigeon-breeder's disease in a child with selective IgA deficiency. Pediatr Int. 2003;45(2):216–8. PMID: 12709156
19. Toprak E, Ogus C, Ozdemir T. Extrinsic allergic alveolitis-pigeon breeder's lung. Arch Lung. 2004;1:48–50
20. Kupeli E, Karnak D, Kayacan O, et al. Clues for the differential diagnosis of hypersensitivity pneumonitis as an expectant variant of diffuse parenchymal lung disease. Postgrad Med J. 2004;80:339–45. doi: 10.1136/pgmj.2003.015669
21. Nacar N, Kiper N, Dogru D, et al. Pigeon breeder's disease in a child treated with inhaled steroid. Cocuk Sagligi Hast Derg. 2005;48:330–3
22. Akgun M, Mirici A, Meral M, et al. A hypersensitivity pneumonitis case complicated with acute respiratory distress syndrome. Respir Med. 2005;99:1195–7. doi: 10.1016/j.rmed.2004.12.008
23. Gocmen S, Saka D, Cimen F, et al. Farmer's lung or miliary tuberculosis? Respir Dis. 2005;16:188–92
24. Ceviz N, Kaynar H, Olgun H, et al. Pigeon breeder's lung in childhood: is family screening necessary? Pediatr Pulmonol. 2006;41(3):279–82. doi: 10.1002/ppul.20355. PMID: 16400661
25. Islamoglu AF, Araz C, Ekiz K, et al. Acute respiratory insufficiency of hypersensitivity pneumonic origin: bird–feeder disease. Erciyes Med J. 2009;31(4):359
26. Kupeli E, Karnak D, Sak S, et al. Hazards of the 'hard cash': hypersensitivity pneumonitis. Can Respir J. 2010;17(5):e102–5. doi: 10.1155/2010/603246. PMID: 23038004
27. Gayaf M, Karasu I, Cakan A, et al. Pigeon breeder's disease (hypersensitivity pneumonitis). Solunum. 2010;12(1):47–51
28. Gungor S, Bilgin S, Akbaba B, et al. Bird breeder's disease: report of two cases. Tuberk Toraks. 2011;59(3):316–17
29. Kantarci M, Arslan B, Karacan M, et al. Pigeon breeder's disease: multi-detector row computed tomography imaging. Eurasian J Med. 2011;43(1):66. doi: 10.5152/eajm.2011.14
30. Erden ES, Isaogullari N, Demirkose M, et al. Pigeon fancier's lung disease. Selcuk Med J. 2014;30(2):74–5
31. Ahat CD, Ekici A, Bulcun E, et al. Güvercin besleyenlerde iyi tanımlanmış bir hastalık olan hipersensitivite pnömonisi. Solunum Hastaliklari Dergisi. 2013;24(1):25–8
32. Ozmen S, Dogru M, Duman H, et al. Bird fancier's lung disease in children: a single center experience. Turk Arch Pediatr. 2013;48(4):327–31. doi: 10.4274/tpa.1169
33. Bahceci ES, Nacaroglu HT, Unsal Kalkiner CS, et al. Hypersensitivity pneumonia in a schoolchild admitted to the hospital's asthma clinic. Turk J Pediatr. 2015;57(5):529–32
34. Aydemir Y, Gungen AC, Coban H. Hypersensitivity pneumonitis caused by the broom grass (Calluna vulgaris). Respir Med Case Rep. 2015;15:135–7. doi: 10.1016/j.rmcr.2015.03.001
35. Bulcun E, Ekici A, Ekici M, et al. A case report: hypersensitivity pneumonia. Turk J Clin Lab. 2016;7(1):26–8
36. Esenboga S, Emiralioglu N, Cagdas D, et al. Diagnosis of interstitial lung disease caused by possible hypersensitivity pneumonitis in a child: think CGD. J Clin Immunol. 2017;37:269–72. doi: 10.1007/s10875-017-0375-4
37. Bekci TT, Calik M, Yalcin B. Hypersensitivity pneumonitis caused by oyster mushrooms: a case of mushroom worker's lung. Res Case Rep. 2018;7(1):1–4. doi: 10.1155/2018/6804037
38. Comlek S. Chemotherapy-induced pneumonitis associated with transient CA 15-3 elevation. J Oncol Pharm Pract. 2020;26(7): 1750–3. doi: 10.1177/1078155219888492
39. Cimrin AH, Goksel O, Demirel YS. General aspects of hypersensitivity pneumonitis in Turkey. Tuberk Toraks. 2010;58(3):242–51
40. Ozsezen B, Ozcan HN, Emiralioglu N, et al. Clinical radiological and pathological staging of children with hypersensitivity pneumonitis. Pediatr Pulmonol. 2022;57(10):2344–55. doi: 10.1002/ppul.26047
41. Öztürk NAA, Coşkun F, Yurttaş A, et al. Systemic corticosteroid treatment response in hypersensitivity pneumonitis: a single center experience. Ege J Med. 2022;61(4):524–9
42. Küpeli E, Karnak D. Hypersensitivity pneumonitis. Tuberk Toraks. 2011;59(2):194–204
43. Vašáková M, Morell F, Walsh S, et al. Hypersensitivity pneumonitis: perspectives in diagnosis and management. Am J Respir Crit Care Med. 2017;196(6):680–9. doi: 10.1164/rccm.201611-2201pp
44. Fink JN, Ortega HG, Reynolds HY, et al. Needs and opportunities for research in hypersensitivity pneumonitis. Am J Respir Crit Care Med. 2005;171(7):792–8. doi: 10.1164/rccm.200409-1205ws

45. Vašáková M, Selman M, Morell F, et al. Hypersensitivity pneumonitis: current concepts of pathogenesis and potential targets for treatment. Am J Respir Crit Care Med. 2019;200(3):301–8. doi: 10.1164/rccm.201811-2092pp
46. Saltoun CA, Harris KE, Mathisen TL, et al. Hypersensitivity pneumonitis resulting from community exposure to Canada goose droppings: when an external environmental antigen becomes an indoor environmental antigen. Ann Allergy Asthma Immunol. 2000;84(1):84–6. doi: 10.1016/s1081-1206(10)62431-6
47. Koschel D, Wittstruck H, Renck T, et al. Presenting features of feather duvet lung. Int Arch Allergy Immunol. 2010;152(3):264–70. doi: 10.1159/000283037
48. Santos V, Martins N, Sousa C, et al. Hypersensitivity pneumonitis: main features characterization in a Portuguese cohort. Pulmonology. 2020;26(3):130–7. doi: 10.1016/j.pulmoe.2019.09.004
49. Zo S, Chung MP, Yoo HY, et al. Clinical characteristics and outcomes of hypersensitivity pneumonitis in South Korea. Ther Adv Respir Dis. 2023;17:1–10. doi: 10.1177/17534666231212304
50. Raghu G, Remy-Jardin M, Myers JL, et al. Diagnosis of idiopathic pulmonary fibrosis: an official ATS/ERS/JRS/ALAT clinical practice guideline. Am J Respir Crit Care Med. 2018;198(5):e44–e68. doi: 10.1164/rccm.201807-1255st
51. American Thoracic Society, European Respiratory Society. Idiopathic pulmonary fibrosis: diagnosis and treatment: international consensus statement. Am J Respir Crit Care Med. 2000;161(2):646–64. doi: 10.1164/ajrccm.161.2.ats3-00
52. Rival G, Manzoni P, Lacasse Y, et al. High-resolution CT predictors of hypersensitivity pneumonitis. Sarcoidosis Vasc Diffuse Lung Dis. 2016;33(2):117–23